Appendix E6

Chemical Properties, Toxicity, and Fate and Transport

CONTENTS

Appendix E6

Chemical Properties, Toxicity, and Fate and Transport

E6-1 EXPLOSIVES

E6-1.1.1 1,3-Dinitrobenzene CAS 99-65-0

Chemical Uses and Properties. Dinitrobenzene (DNB) is an impurity of TNT. It occurs as a pale yellow or white crystalline solid (HSDB 2000). It has been found in soils of OB/OD areas and waste waters associated with munitions factories (Layton et al. 1987). Some important chemical properties are summarized from Layton et al. (1987) and HSDB (2000) below:

- Henry's Law Constant 2.33×10^{-6} atm-m^3 mol^{-1}
- Log Kow 1.49
- Koc 210
- Aqueous Solubility 180 mg/L
- Molecular Weight 168.12

E6-1.1.1.1 Fate and Transport. The important environmental fate processes for 1,3-DNB are uncertain. Simmons and Zepp (1986) determined that the average annual rate constant for the direct photolysis of 1,3-DNB in distilled water was 0.03 $(day)^{-1}$ at 40°N latitude. Spanggord et al. (1980) noted a 50% loss of the 1,3-DNB component of condensate water after 12 days of exposure to sunlight. Therefore, photolysis may represent a significant fate process. Volatilization is probably not an important loss mechanism for 1,3-DNB. Physical transport of 1,3-DNB from aqueous systems is believed to be unimportant because of its low vapor pressure of $1.31*10^{-4}$ at 25°C (Maksimov 1963).

Although adsorption to clays can occur, 1,3-DNB is not expected to remain in most soils for extended periods of time, and leaching will occur (HSDB 2000). Hydrolysis and volatilization from soils are not expected to be major fate processes (Spanggord et al. 1980). Microbes have been observed to degrade DNB into 3-nitroaniline under both aerobic and anaerobic conditions with a half-life of less than two weeks (Hallas and Alexander 1983); reduction to aromatic amines occurs under anaerobic conditions (HSDB 2000). Conversely, however, other research indicates no biodegradation. Bioaccumulation is probably not an important transport mechanism for 1,3-DNB. Isnard and Lambert (1988) determined the bioconcentration factor (BCF) for 1,3-DNB in fish to be 4.7. Other estimates provide a BCF of 6 for fish (Layton et al. 1987) and a BCF of 0.9 for fish (HSDB 2000). The estimated bioaccumulation factor (BAF) for uptake from soil by plants is 4 on a wet-weight basis, and the BAF for cattle for uptake from diet into fat is 0.0022 (Layton et al. 1987).

E6-1.1.1.2 Toxicity. Most orally administered 1,3-DNB is absorbed by rats and rabbits (Parke 1961; Nystrom and Ricket 1987). Single and oral doses in rodents have been associated with encephalopathy (Philbert et al. 1987). Numerous investigators have also studied the adverse effects of 1,3-DNB on male reproductive function (USEPA 1991b). These effects include Sertoli cell damage, damage to the seminiferous epithelium, reduction in late pachytene spermatocytes, decreased testicular weights, impairments in sperm morphology and motility, and reduced fertility. The lowest acute and subchronic doses associated with these effects were 15 mg/kg and 0.54 mg/kg/day, respectively.

Adequate chronic data and information on effects about the female reproductive system were not available (USACE 1993). DNB is readily absorbed through the skin. A volunteer wearing latex gloves immersed for 45 minutes in 5,000 ppm of DNB exhibited increased methemoglobin levels (Ishihara et al. 1976). A similar response has been observed in cats dermally exposed to DNB (White and Hay 1901). The primary routes of metabolism involve reduction of the nitro groups and oxidation of the aromatic ring to a phenol, and data suggest that excretion is predominantly by the urinary tract (Layton et al. 1987). Other adverse effects associated with exposure to 1,3-DNB are decreased growth rate, weight loss, anemia, methemoglobinemia, and cyanosis (HSDB 2000).

1,3 DNB is a Group D carcinogen, which means that it is not classifiable as to human carcinogenicity due to the lack of human and animal data. The Federal drinking water guideline is 1 ug/L (HSDB 2000).

E6-1.1.2 2,4-Dinitrotoluene CAS 121-14-2

E6-1.1.2.1 Chemical Uses and Properties. 2,4 Dinitrotoluene (2,4-DNT) is an impurity of TNT and is the primary component of military-grade DNT, which is used in some propellent formulations. It occurs as yellow or orange crystals (HSDB 2000). It has been found in soils of OB/OD areas (Layton et al. 1987). Some important chemical properties are summarized from Layton et al. (1987) and HSDB (2000) below:

- Henry's Law Constant 1.3×10^{-7} atm-m^3 mol^{-1}
- Log Kow 1.98
- Koc 260 – 360
- Aqueous Solubility 270 – 273 mg/L
- Molecular Weight 182.14

E6-1.1.2.2 Fate and Transport. Photolysis and sorption are the most probable fate processes for 2,4-DNT. Photolysis in solution may be a highly probable fate process for 2,4-DNT (Callahan et al. 1979), whereas photoreduction appears to be a less significant degradation pathway. The photolysis half-life of 2,4-DNT in pure water is 42 hours (Spanggord et al. 1980), and the half-life in natural water is much shorter. Simmons and Zepp (1986) found that dissolved or suspended humic substances greatly enhance (10 to 17 times). Spanggord et al. (1980) concluded that adsorption to sediment was not an important environmental process for 2,4-DNT. Leaching may occur and, therefore, adsorption to soil and sediment may be very minor (Burrows et al. 1989).

A vapor pressure of 1.47×10^{-4} mm Hg at 2°C suggests that if released to air, 2,4-DNT will exist solely as a vapor in the ambient atmosphere (HSDB 2000). Atmospheric degradation with a half-life of 75 days occurs by reaction with hydroxyl radicals produced by photochemical reactions (HSDB 2000).

Studies on 2,4-DNT soil adsorption have been somewhat conflicting. Based on the ability of polynitroaromatic compounds to form very stable charge-transfer complexes with more highly electronegative aromatic compounds (Hall and Pornaski 1970), 2,4-DNT should be strongly adsorbed by both humus and clay. Also, the log octanol/water partition coefficient (K_{ow}) of 2.01, calculated by the method of Tute (1971), is sufficiently large to indicate significant adsorption of 2,4-DNT by humus (Callahan et al. 1979). 2,4-DNT is not expected to volatilize from moist or dry soil surfaces given the Henry's Law constant and vapor pressure (HSDB 2000).

Studies of the biodegradation of 2,4-DNT have produced conflicting results. In general, nitrotoluenes are resistant to biodegradation by soil microorganisms (Alexander and Lustigman 1966). It has been reported that dinitrotoluenes decompose very slowly in a reservoir (Galuzova 1963). Bioaccumulation may not be an important process for 2,4-DNT. The contention is supported by analogy with nitrobenzene and relatively low log K_{ow} of 2.01 for 2,4-DNT (Callahan et al. 1979).

Dames and Moore (1991) determined a low bioconcentration factor of 1.06 for 2,4-DNT (USACE 1993). Other sources indicate BCFs as high as 25 to 78 (Layton et al. 1987). BCFs for fish estimated from log Kow values are 15 (Layton et al. 1987). Uptake by plants from soil and mammals from their diet is estimated to be low. BAFs for plants and cattle are 4 and 0.0027, respectively.

E6-1.1.2.3 Toxicity. The oral absorption of 2,4-DNT is highly dependent on species and strain; although an accurate determination of the amount absorbed cannot be made, most experimental animals excrete over half of orally-administered radioactivity in the urine of bile (USEPA 1992). Rats are more sensitive to single dose than mice. In both species, the clinical signs of overdose include ataxia and cyanosis (Lee et al. 1975). Dogs are more sensitive than rodents in subacute studies and show signs of decreased body weight gain and spermatogenesis and neuromuscular incoordination and paralysis at doses as low as 25 mg/kg/day (Lee et al. 1978). This order of species sensitivity also occurs in two-year studies. The primary target organs, affected in all dogs exposed to 10 mg/kg/day and one dog treated with 1.5 mg/kg/day, were the nervous system, erythrocytes, and biliary tract (Ellis et al. 1985). The major effect, central nervous system toxicity, included vacuolization and cerebellar damage. Chronically treated male rats had seminiferous tubule atrophy at 3.9 mg'/kg/day (Lee et al. 1985) and similarly treated micc showed weight gain decrements at doses of 14 mg/kg/day and higher (Hong et al. 1985)(USACE 1993). 2,4-DNT is considered a possible human carcinogen (Group 2B), where there is inadequate evidence to predict carcinogenicity in humans, but there is sufficient evidence that 2,4-DNT is carcinogenic in experimental animals.

E6-1.1.3 2,6-Dinitrotoluene CAS 118-96-7

E6-1.1.3.1 Chemical Uses and Properties. 2,6-Dinitrotoluene (2,6-DNT) occurs as yellow crystals and is a constituent in military grade TNT. It has been found in soils where demililitarization activities have occurred (Layton et al. 1987). It may burn, but does not ignite readily (HSDB 2000). Some important chemical properties estimated by Layton et al. (1987) and HSDB (2000) are summarized below:

- Hentry's Law Constant 9.26×10^{-8} atm-m^3 mol^{-1}
- Log Kow 1.9 – 2.10
- Koc 19 – 72
- Aqueous Solubility 910 mg/L
- Molecular Weight 182.14

E6-1.1.3.2 Fate and Transport. Photolysis, sorption, and biodegradation are the most probable fate processes for 2,6-DNT. Photolysis in solution may be a highly probable fate process for 2,6-DNT (Callahan et al. 1979). The photolysis half-life of 2,6-DNT in pure water is 42 hours (Spanggord et al. 1980). Simmons and Zepp (1986) found that dissolved or suspended humic substances greatly enhance (10 to 17 times) indirect photolysis of 2,6-DNT (USPHS 1989). It has been reported that

dinitrotoluenes decompose very slowly in a reservoir (Galuzova 1963). More recently, Spanggord et al. (1980) observed biodegradation of DNTs in an aerobic environment with a half-life of less than one hour.

If released to air, the vapor pressure of 5.67 x 10^{-4} mm Hg at 25°C indicates that 2,6-DNT will exist solely as a vapor in ambient atmosphere. Atmospheric degradation with a half-life of 75 days will occur by reaction with photochemically-produced hydroxyl radicals (HSDB 2000). 2,6-DNT is not expected to volatilize from moist or dry soil (HSDB 2000).

The ability of polynitroaromatic compounds to form highly stable charge-transfer complexes with more highly electronegative aromatic compunds (Hall and Poranski 1970) indicates that 2,6-DNT should be strongly adsorbed by both humus and clay. Also, its log octanol-water partition coefficient (K_{ow}) of 2.05, calculated by the method of Tute (1971), is sufficiently large to indicate significant adsorption of 2,6-DNT by humus (Callahan et al. 1979). Leaching may occur and, therefore, adsorption to soil and sediment may be very minor (Burrows et al. 1989). However, measured Koc values ranging from 19 to 72 suggest high mobility and little adsorption to soils (HSDB 2000). In the absence of sunlight and oxygen, any losses of DNTs would apparently be dependent on biodegradation (USPHS 1989). Reported half-lives for biodegradation in soil range from 73 to 92 days (HSDB 2000).

Bioaccumulation may not be an important process for 2,6-DNT. This contention is supported by 2,6-DNT's relatively low log K_{ow} of 2.05 (Callahan et al. 1979) (USACE 1993). A measured BCF of 11 (HSDB 2000) provides further evidence for a lack of bioconcentration in aquatic species.

E6-1.1.3.3 Toxicity. The adverse effects associated with 2,6-DNT exposure include skin irritation, nervous system effects, methemoglobinemia, anemia, leukopenia, and liver necrosis, where liver injury may be more common than cyanosis. In rodents, most orally administered 2,6-DNT is excreted in the urine or feces, indicating extensive adsorption (USEPA 1992). Clinical signs of acute intoxication include cyanosis and ataxia. After subacute, subchronic, exposure, mice, rats, and dogs show decreased body weight gain, anemia with compensatory hematopoiesis, and reduction in spermatogenesis (USEPA 1992). Dogs also showed nervous system signs, including neuromuscular incoordination and lethargy. The LOELs for adverse effects in subchronic dog, rat, and mice studies were 20, 35, and 51 mg/kg/day, respectively. The greater covalent hepatic binding of 2,6-DNT, and its greater carcinogenic potential, compared to the 2,4-isomer, suggest that it is the more toxic of the two isomers (Ricket et al. 1983; Leonard et al. 1987) (USACE 1993). 2,6-DNT is considered a possible human carcinogen (Group 2B), where there is inadequate evidence to predict carcinogenicity in humans, but there is sufficient evidence that 2,4-DNT is carcinogenic in experimental animals.

E6-1.1.4 HMX (Cyclotetramethylenetetranitramine) CAS 2691-41-0

E6-1.1.4.1 Chemical Uses and Properties. HMX is one of the most powerful and widely used military explosives. Its uses include a booster charge in mixtures with TNT, an oxidizer in solid rocket and gun propellants, and as an explosive charge (DOA 1984). Soil and groundwater contamination have been associated with manufacturing, waste discharge, testing and training, demilitarization of ordnance, and OB/OD operations (Hawari 2000). HMX is a nonaromatic cyclic nitramine. Some important chemical properties are summarized from Layton et al. (1987) and HSDB (2000) below:

- Henry's Law Constant
- Log Kow 0.59
- Koc 130 – 670

- Aqueous Solubility 2.6 – 5.0 mg/L
- Molecular Weight 296.20

E6-1.1.4.2 Fate and Transport. Photolysis will degrade HMX in shallow surface water. HMX is practically insoluble in water and is nonhydroscopic. Its solubility in other solvents is comparable to that of RDX. HMX is considered extremely stable for a high explosive. HMX absorbs the greatest number of photons at wavelengths below 290 nanometers (nm) (Maycock et al. 1969); however, HMX degrades in natural light by absorbing light between 290 and 370 nm (Spanggord et al. 1983; Maycock et al. 1969; Smetana and Bulusu 1977). The photolytic decay rates for HMX range from 0.0036 to 0.4 (Zepp and Cline 1977), and the corresponding half-lives range from 1.7 to 192 days. Composting has been shown to be an effective means of bioremediating soils contaminated with HMX (Bruns-Nagel et al.2000). Once the cyclic HMX undergoes a molecular change to the ring sturcture, the ring collapses to produce small nitrogen and carbon products (Hawari 2000). HMX is more stable and not as amenable to biodegradation as is RDX (Hawari 2000). The information regarding HMX's tendency to sorb to soils is inconclusive. Data indicate adsorption of HMX is slight; however, leaching could be important (Burrows et al. 1989).

Bioaccumulation is probably not an important transport mechanism for HMX. Studies of the effects of oral doses of HMX (500 mg/kg) on mice and rats found that the chemical reached a peak concentration of 6 to 10 g/mL in plasma within six hours. In contrast, intravenous doses of HMX (2 mg/kg) reached a peak concentration of 0.5 to 1 g/mL within an hour (Wilson 1985). Aquatic BCFs can be calculated for explosives from empirical relationships. Burrows et al. (1989) calculated the HMX BCF in fish to be 0.49, which indicates only a very low tendency for HMX to accumulate in aquatic life (USACE 1993).

E6-1.1.4.3 Toxicity. The limited data on the gastrointestinal absorption of HMX suggest that uptake by the systemic circulation is poor (Henderson 1985). Clinical signs of intoxication in mice exposed to a single high dose included hyperkinesia, ataxia, and sedation (Cuthbert et al. 1985). Mice exposed subchronically to dietary concentrations corresponding to 200 to 750 mg/kg/day typically died with no clinical or histopathological signs of toxicity (Everett and Maddock 1985). Lower dose equivalents were not associated with any signs of toxicity or mortality. In general, this study was not adequately designed for determination of a no-observed-adverse-effect level (NOAEL) or lowest-observed-adverse-effect level (LOAEL). Adverse effects in rats exposed to dietary concentrations corresponding to 150 mg/kg/day or higher included irreversible body weight gain decrements and histological evidence of liver and kidney damage (Everett et al. 1985). Neither a chronic toxicity nor a reproductive/developmental database were available (USACE 1993). HMX is considered Class D (not classifiable as to human carcinogenicity) (HSDB 2000). A water quality criterion for the protection of freshwater aquatic life of 750 ug/L was proposed, and the Federal drinking water guideline is 400 ug/L (HSDB 2000).

E6-1.1.5 Nitrobenzene CAS 98-95-3

E6-1.1.5.1 Chemical Uses and Properties. Nitrobenzene is used in manufacturing. It is also produced in the atmosphere from the photochemical reaction of benzene with oxides of nitrogen. Some important chemical properties are summarized from Layton et al. (1987) and HSDB (2000) below:

- Henry's Law Constant 2.4×10^{-5} atm-m^3 mol^{-1}
- Log Kow 1.84
- Koc 130.6 - 370

- Aqueous Solubility
- Molecular Weight 123.11

E6-1.1.5.2 Fate and Transport. The movement of nitrobenzene in soil, water, and air is dominated by its water solubility (1,900 parts per million [ppm]) (Verschueren 1983), moderate volatility, low octanol-water partition coefficient, and soil/sediment sorption coefficient. There is no known mechanism of hydrolysis of nitrobenzene; however, photolysis and biodegradation are significant pathways (Callahan et al. 1979; Mabey et al. 1983). Photochemical oxidation of nitrobenzene by H_2O_2 yields p-, o-, and m-nitrophenols (Draper and Crosby 1984) with an estimated half-time of 250 days (Dorfman and Adams 1973). Direct photolysis, measured by Zepp and Scholtzhauer (1983), has a half-time of 2.5 to more than 6 days near the surface of bodies of water in the vicinity of 40°N latitude.

Conflicting results on the biodegradation of nitrobenzene are present. It has been shown to be almost completely removed from natural waters or in various sewage treatment processes in some studies. Other studies show the majority of nitrobenzene removed, but at a much slower rate. A few studies have shown nitrobenzene to be either highly resistant to degradation or to be inhibited from biodegradation of other compounds in the medium (USACE 1993).

Jury et al. (1984) classified nitrobenzene as intermediately mobile, but noted that its loss from soil would be enhanced by evaporation of water. Volatilization from moist soil surfaces may be an important fate process (HSDB 2000). Sediment sorption and bioconcentration into aquatic and terrestrial animals will be negligible, whereas plant uptake might be expected in terrestrial (McFarlane et al. 1987a, 1987b), but not aquatic systems (Geyer et al. 1984). Leaching through soil may be significant.

Nitrobenzene has a vapor pressure of 0.245 mm Hg at 25°C. It is expected to degrade in the atmosphere by photolysis and reaction with hydroxl radicals with a half-life of 115 days (HSDB 2000).

In air, nitrobenzene exists as a vapor (vapor pressure 0.245 mm Hg at 25°C), and degrades by reaction with hydroxyl radicals with a half-life estimated to be about 115 days (HSDB 2000).

Bioaccumulation does not appear to be a major fate process. Nitrobenzene binds to the roots of soybeans, but less than 1.5% was translocated to the plant shoots; green algae exhibited a BCF of 24 (HSDB 2000). BCFs for fish range from 1.6 to 15 (HSDB 2000).

E6-1.1.5.3 Toxicity. An LD_{50} of 600 mg/kg in rats was reported by Smyth et al. (1969), and an oral lethal dose for a rabbit was 200 mg/kg (HSDB 2000). One study was found on the effects to the reproductive system from nitrobenzene. An acute systemic study in rats indicated that the testis is sensitive to the toxic effects of nitrobenzene. Typical signs included testicular degeneration and transiently decreased sperm production following a single oral dose of 300 mg/kg (Levine et al. 1988) (USACE 1993). Male rats were administered a single oral dose between 50 and 450 mg/kg; rats exhibited hepatic lesions at various doses, and testicular lesions at doses of 300 mg/kg (HSDB 2000). Inhalation studies indicate exposure produces moderate bronchiolar hyperplasia, reduced maternal weight gain, lesions of nose, liver, testis, and lung, degeneration of olfactory epithelium, but no effect on fetal reproductive endpoints of rats and mice (HSDB 2000). A multigenerational study showed male reproductive tract effects at concentrations of 40 ppm in air (HSDB 2000).

Nitrobenzene is considered a Group D (not classifiable as to human carcinogenicity) compound by EPA, but a Group 2B (possibly carcinogenic to humans) by the World Health Organization (HSDB 2000), where the evidence for carcinogenic effects in humans and animals is lacking. Exposure causes methemoglobin formation and cyanosis, weakness, ataxia, dyspnea, and tachycardia

(HSDB 2000). Liver damage is observed following repeated exposure. Nitrobenzene is readily dermally absorbed; through human skin the absorption rate is a high as 2 mg/m^2-h (HSDB 2000).

E6-1.1.6 RDX (Hexahydro-1,3,5-Trinitro-1,3,5-Triazine) CAS 121-82-4

E6-1.1.6.1 Chemical Uses and Properties. RDX is a white, crystaline powder and is one of the most powerful and widely used military explosives. RDX is a nonaromatic cyclic nitramine. RDX can be released to the environment during manufacturing or during explosive use (HSDB 2000). Some important chemical properties are summarized from HSDB (2000) below:

- Henry's Law Constant 6.3×10^{-8} atm-m^3 mol^{-1}
- Log Kow 0.87
- Koc 42 - 167
- Aqueous Solubility 59.8 @ 25°C
- Molecular Weight 222.26

RDX is a high explosive is used as base charge for detonators and as an ingredient of bursting charges and plastic explosives by the military. The melting point of RDX ranges between 205–207° C. High explosives like RDX decompose by detonation. This detonation occurs almost instantaneously and is violent. The explosion may be initiated by sudden shock, high temperature or a combination of the two (Spectrum 2000). Areas contaminated with RDX can be found at a few of the ordnance sites along the Idaho National Engineering and Environmental Laboratory (INEEL). Many of these areas were used for explosive testing and experimentation, and still contain RDX chunks as large as a baseball. However, most chunks found at these sites were under an inch in size.

E6-1.1.6.2 Fate and Transport. Transformation of RDX in the environment occurs most rapidly by photolysis. Spanggord et al. (1980) measured the degradation of RDX in distilled and filter-sterilized natural water in sunlight. They calculated a half-life of 13 days from their measurements of RDX disappearance from distilled water, 14 days in Holston River water, and 9 days in Searsville Pond water during cloudy January weather.

Hydrolysis should be a significant environmental fate of RDX only in alkaline waters (Hoffsommer and Rosen 1973), but should not occur in freshwater. Measurements of soil-water partition coefficient and organic-carbon partition coefficient (Tucker et al. 1985; Spanggord et al. 1980; and Sikka et al. 1980) indicate that RDX does not strongly adsorb to soils and sediments.

Biological processes can play a role in determining the fate of RDX. Microbial reduction of RDX usually takes place under anaerobic conditions. McCormick et al. (1981) found that RDX disappeared from nutrient broth cultures within four days of anaerobic incubation, but found no loss from aerobic cultures or controls. Spanggord et al. (1980) found that aqueous solutions of RDX were aerobically degraded over 16 days from 10 ppm to 4 ppm by adapted microbes in Holston River water after a 20-day lag time. They reported that RDX does not undergo biotransformation without added nutrients or adapted bacteria. Biodegradation in soil is expected to be complete within 24 days under anerobic conditions, but RDX is resistant to biodegradation in soil under aerobic conditions (HSDB 2000).

Studies by Cataldo et al. (1990) indicate that RDX bioaccumulation from soils by plants is a significant route of transport. Cataldo et al. (1990) amended soils (i.e., Burbank, Palouse, and Cinebar)

with 10 ppm RDX and found that bush bean removed 55% of the RDX in Burbank soil, 37% from Palouse, and 11% from Cinebar. In addition, blando brome grass was found to remove 45% the RDX amended to Burbank soil. From the Cataldo data, a calculated BCF factor for *Blando brome* in Burbank soils is 91. Cataldo et al. (1990) also determined that partitioning of RDX occurs in plants; approximately 20% is stored in the roots and 80% among the shoots, fruits, leaves, and seeds. These data suggest that bioaccumulation of RDX by plants presents a potential food-chain contamination route for RDX to higher trophic order organisms (USACE 1993).

E6-1.1.6.3 Toxicity. Results of rodent studies consistently indicate that orally administered RDX, although adequately absorbed, has little bioaccumulation potential (Schneider et al. 1977, 1978). Confirming the clinical signs observed in occupationally exposed individuals, rats, mice, and dogs exposed to high single oral doses show central nervous toxicity, including labored breathing and convulsions (USEPA 1988). The expression of toxicity depends on the particle size of the RDX preparation, with fine powders showing the greatest effect (Schneider et al. 1977). After prolonged exposure, the order of species sensitivity is dog>rat>mouse. Based on chronic dietary studies, the rat LOAEL (associated with prostate inflammation) was 1.5 mg/kg/day (Levine et al. 1983a) and the mouse LOAEL (associated with testicular atrophy) was 35 mg/kg/day. These doses resulted in hyperirritability, weight loss, convulsions, and severe gastrointestinal irritation (Con Oettingen et al. 1949). Treatment of dogs for 90 days with 10 mg/kg/day RDX had no observable toxic effect, except for periods of emesis (Hart 1974) (USACE 1993).

RDX is listed as a possible human carcinogen (Class C) on the basis of carcinogenic effects in mice (HSDB 2000). The federal drinking water guideline is 2 ug/L (HSDB 2000).

E6-1.1.7 Tetryl (2,4,6-Tetryl-Trinitophenylmethylnitramine) CAS 479-45-8

E6-1.1.7.1 Chemical Uses and Properties. Tetryl is a colorless to yellow crystalline solid that was used as a booster explosive, which is the explosive ignited by a detonation charge which in turn ignites a bursting charge (HSDB 2000). Some important chemical properties are summarized from Layton et al. (1987) and HSDB (2000) below:

- Henry's Law Constant 1.0×10^{-11} atm-m^3 mol^{-1}
- Log Kow 2
- Koc 406
- Aqueous Solubility 75 mg/L @ 20°C
- Molecular Weight 287.15

E6-1.1.7.2 Fate and Transport. Biodegradation and photolysis are the only significant degradation processes of tetryl. Kayser et al. (1984) reported that N-methylpicramide was the major detectable photoproduct of tetryl in distilled water exposed to laboratory "room light." Kayser et al. (1984) also stated that the photolysis rate is at least one more order of magnitude than the hydrolysis rate. Photolysis of tetryl in sunlight proceeds more slowly than the photolysis of the nitramines, requiring 20 days for completion. Hydrolysis has been demonstrated to occur to tetryl; however, this process is slow. Kayser et al. (1984) have reported a half-life (extrapolated) for this reaction of 302 $\pm$ 76 days at 20°C and a pH of 6.8. Kayser et al. (1984) also reported that the rate of hydrolysis increases with both pH and temperature. Moderate leaching from soil is expected to occur (HSDB 2000).

Volatilization is considered to have an insignificant role in decreasing the concentration of explosives in environmental media (Burrows et al. 1989). The low Henry's Law coefficient suggests tetryl will not volatilize from moist soils, and a calculated vapor pressure of 1 x 10-8 mm Hg indicate that tetryl in ambient air will exist in the particulate and not vapor form.

Adsorption to sediment is not an important environmental process for this explosive in surface water (Spanggord et al. 1980). No data on adsorption to soils was located, however, tetryl's low organic carbon partition coefficient (K_{oc}=44.67) indicates adsorption to soils is not a significant process. This is probably not an important transport process.

Based on a report by Trabalka and Garten (1982) on cutoff values for log octanol/water partition coefficient (K_{ow}) and solubility, tetryl does not accumulate in mammals or birds. This contention is supported by Burrows et al. (1989), who reported a tetryl bioconcentration factor (BCF) of 6.31 for fish and a very low BCF of 0.0023 for fat feed beef (USACE 1993). Other estimates of BCF for tetryl are as high as 54 (HSDB 2000).

E6-1.1.7.3 Toxicity. Tetryl exposure results in dermatitis, as well as conjuctivitis, keratitis, and iridocyclitis of the eyes, acute irritation of nasal mucus membranes. Chronic exposure can result in appetite loss, abdominal pain, vomiting, weight loss, chronic hepatitis, central nervous system effects, and anemia (HSDB 2000). Based on limited analyses of tetryl metabolites in the urine of rabbits, gastrointestinal absorption may be very slow (Zambrano and Mandovano 1956). Short-term oral dosing with 250 mg/kg/day has been associated with degenerative changes in kidneys and spleen, and focal necrosis of the liver (Parmeggiani et al. 1956; Guarino and Zambrano 1957). Only two subchronic studies are available (Daniele 1964; Fati and Daniele 1965). Exposure of rabbits to 125 mg/kg/day for up to nine months resulted in hepatocyte swelling and vascular congestion, swelling and degeneration of the renal tubules, congested spleen, and lymphatic atrophy. The severity of the effects of this dose equivalent (corresponding to the only treatment level used in the study) suggest frank toxicity. No chronic toxicity or reproductive/developmental toxicity data were available (USACE 1993).

E6-1.1.8 1,3,5-Trinitrobenzene CAS 99-35-4

E6-1.1.8.1 Chemical Uses and Properties. 1,3,5-Trinitrobenzene (TNB) is a by-product of TNT synthesis, as well as a breakdown product of TNT photolysis (Layton et al. 1987). Some important chemical properties are summarized from Layton et al. (1987) below:

- Henry's Law Constant 6.6 x 10 -7 atm-m^3 mol^{-1}
- Log Kow 1.18
- Koc 77
- Aqueous Solubility 330
- Molecular Weight 213.12

E6-1.1.8.2 Fate and Transport. The major environmental fate processes for 1,3,5-TNB are not well documented. TNB is biodegraded to 3,5-dinitroaniline (HSDB 2000). Studies by Burlinson (1980, 1973a, 1973b) suggest that photolysis may not be a significant transformation process. Physical transport of 1,3,5-TNB from aqueous systems is believed to be unimportant because of its low vapor pressure, $3.03*10^{-6}$ mm Hg at 25°C (Burrows et al. 1989). In addition, its Henry's law constant of 6.6×10^{-7} at 25°C indicates little tendency for volatilization from water. Sorption of 1,3,5-TNB from aqueous systems

is believed to be unimportant because of its low sediment adsorption coefficient of 19.95 (Burrows et al. 1989). Hydrolysis is also not expected to be a major fate process (HSDB 2000).

Isnard and Lambert (1988) report a BCF of 2.65 in fish, which indicates only a very slight tendency for 1,3,5-TNB to accumulate in aquatic life. Trabalka and Garten (1982) reported that chemicals with log K_{ow} values less than 3.5 or solubilities greater than 10 mg/L do not accumulate in mammals or birds. Therefore, 1,3,5-TNB, which has a K_{ow} of 15.14 and a solubility value of 385 mg/L, would not tend to accumulate in animal receptors in significantly high concentrations (USACE 1993).

E6-1.1.8.3 Toxicity. Pharmacokinetic data for 1,3,5-TNB are not available. Exposure to a single high dose resulted in cyanosis and central nervous system and respiratory disorders (Korolev et al. 1977). The reported LD_{50} values were 450 mg/kg in rats, 600 mg/kg in mice, and 730 mg/kg in guinea pigs. A single oral dose of 0.4 micromoles/kilogram produced methemoglobinemia in rats (Senczuk et al. 1976). No subchronic, chronic, or reproductive/developmental toxicity data are available for 1,3,5-TNB. In the absence of these data, quantitative risk assessment has been done by analogy to 1,3-dinitrobenzene (Cody et al. 1981; USEPA 1992) (USACE 1993).

E6-1.1.9 2,4,6-Trinitrotoluene CAS 118-96-7

E6-1.1.9.1 Chemical Uses and Properties. TNT is a high explosive compound. It has been found in soils of OB/OD areas and waste waters associated with munitions factories (Layton et al. 1987). Some important chemical properties are summarized HSDB (2000) below:

- Henry's Law Constant 4.57×10^{-7} atm-m^3 mol^{-1}
- Log Kow 1.6
- Koc 1100 - 1900
- Aqueous Solubility 13 0 mg/L @ 20°C
- Molecular Weight 227.13

2,4,6-TNT, a pale yellow, solid organic nitrogen compound used chiefly as an explosive, is prepared by stepwise nitration of toluene. Because 2,4,6-TNT melts at 82° C (178° F) and does not explode below 240° C (464° F), it can be melted in steam-heated vessels and poured into casings. It is relatively insensitive to shock and cannot be exploded without a detonator. For these reasons it is the most favoured chemical explosive, extensively used in munitions and for demolitions (Britannica 2000). Concentrated soil contamination from 2,4,6-TNT can be found in many ordnance areas found at the INEEL. Many of these areas were used for explosive testing and experimentation. Large particles of 2,4,6-TNT can still be found at most of these sites; sizes range from a quarter inch to a half inch in diameter.

E6-1.1.9.2 Fate and Transport. In aqueous environments, 2,4,6-TNT is degraded primarily through photolysis and secondarily through hydrolysis (Spanggord et al. 1981). In general, nitroaromatics resist hydrolysis (HSDB 2000). The photolytic rate is related to increases in pH level and organic matter content (HSDB 2000); estimates of half-lives from natural and distilled water under different conditions of latitude, season, and sunlight or shade ranged from as low as 10 minutes to as high as 730 days (HSDB 2000).

Half-lives in soil have been reported as ranging from 4 to 10 years, with the microbial oxidative decomposition rate varying from 0.05 to 0.017 percent per day (Cataldo et al. 1989). TNT is readily degraded in soils only under anaerobic conditions; microbial transformation leads to the formation of DNTs and other reduction products (HSDB 2000). Photolysis in surface soils results in transformation to trinitrobenzene and trinitrobenzaldehyde. The extent of soil sorption is significant, and positively correlated with both pH and temperature (Kayser and Burlison 1988; Sikka et al. 1980). Soil mobility is thus limited, because of strong adsorption to soil particles (EPA 1990). 2,4,6-TNT has a soil permeability rate of 3.4E-3 cm/hr (RAIS 2000).

Cataldo et al. (1989) observed that the 2,4,6-TNT uptake rate in bush bean, wheat, and blando brome was approximately 60 micrograms/hour/gram fresh weight root. These studies suggest that 2,4,6-TNT becomes incorporated into plants as polar metabolites. Toxic concentrations range from 1 ppm duckweed (Schott and Worthley 1974) to 30 ppm in bean, wheat, and blando brome (Cataldo et al. 1989).

Vapor-phase TNT will react with photochemically produced hydroxyl radicals with a half-life of 110 days (HSDB 2000). The vapor pressure is 1.99×10^{-4} mm Hg at 20°C indicating that TNT will exist in the vapor as opposed to particulate phase; the Henry's Law constant suggests only minimal volatilization from moist soils (HSDB 2000).

The plant uptake factor for 2,4,6-TNT is 1.8 (RAIS 2000). A bioconcentration factor of 19 suggests that bioaccumualation is not a significant fate process (Dames and Moore 1991) (USACE 1993). Other estimates provide BCFs for fish ranging from 9.5 to 2030 (HSDB 2000), where higher concentrations were observed in viscera than muscle.

E6-1.1.9.3 Toxicity. Acute exposure of mammals to high oral doses is associated with tremors, convulsions, lassitude, and red-stained urine (USEPA 1990a). When administered chronically, 2,4,6-TNT adversely affects a variety of organ systems. The predominant effects include methemoglobinemia, anemia, testicular degeneration, nephropathy, and decreased body weight gain (USEPA 1990a). Dogs are the most sensitive common laboratory species to these effects, showing mild hepatocellular changes at dose equivalents of as low as 0.6 mg/kg/day (Levine et al. 1983b). Testicular atrophy and Leydig cell hypertrophy occurred in rats at a subchronic dose of 25 mg/kg/day (Levine et al. 1983a). Testicular atrophy and Leydig cell hypertrophy occurred in rats at a subchronic dose of 25 mg/kg/day (Levine et al. 1983a); however, these results were not replicated in a two-year study using 50 mg/kg/day (Furedi et al. 1984). The results of pharmacokinetic studies indicate extensive absorption in all mammalian species after oral administration (USEPA 1990a) (USACE 1993).

2,4,6-TNT is absorbed through the gastrointestinal tract, skin, and lungs; it is distributed primarily to the liver, kidneys, lungs, and fat, and is excreted mainly in the urine and bile (El-hawari et al. 1981). Metabolism occurs by nitroreduction to amino and hydroxylamino derivatives and by oxidation to benzyl alcohol and benzoic acid derivatives (El-hawari et al. 1981).

In animals, signs of acute toxicity to 2,4,6-TNT include ataxia, tremors, and mild convulsions. Splenic hemosiderosis, leukopenia, thrombocytosis, slight hepatomegaly, and increase in kidney weight occurred in mice fed a dietary level equivalent to 700 mg 2,4,6-TNT/kg/day for 28 days (Levine et al. 1984). Oral LD_{50} values of 660 to 1,320 mg/kg have been reported for rats (Dilley et al. 1982).

The primary target organs for 2,4,6-TNT toxicity in experimental animals following subchronic and chronic oral exposures are (1) liver (hepatocytomegaly and cirrhosis), (2) blood (hemolytic anemia with secondary alterations in the spleen), and (3) testes (degeneration of the germinal epithelium lining

the seminiferous tubules). The LOAEL for hepatotoxicity in dogs was 0.5 mg/kg/day (Levine et al. 1990).

Chronic oral toxicity studies on rats have also demonstrated TNT-induced anemia and hepatotoxicity, as well as adverse effects on the kidney (hypertrophy and nephropathy) and sternal bone marrow fibrosis (Furedi et al. 1984a).

The reference dose (RfD) for chronic oral exposures, 0.0005 mg/kg/day, is based on a LOAEL of 0.5 mg/kg/day for liver effects in dogs (EPA 1991b). The subchronic oral RfD is the same as the chronic RfD and is based on the same study (EPA 1991a). TNT is a Group C (possible human carcinogen), where the evidence for human carcinogenicity is inadequate, and the animal carcinogenicity data are limited. The Federal drinking water guideline is 2 ug/L.

Information on the inhalation toxicity of 2,4,6-TNT is derived mainly from occupational exposure studies, which indicate that the major effects of chronic exposure to 2,4,6-TNT are anemia (decreases in Hgb, Hct, and RBC count), liver dysfunction (increases in serum lactic dehydrogenase, glutamic oxaloacetic transaminase, and bilirubin), and cataracts (equatorial lens opacities) (EPA 1989, 1990). Other reported effects of TNT exposure include dermatitis, leukocytosis, neurological disorders, and nephrotoxicity (Cone 1944; Zakhari and Villaume 1978). An inhalation reference concentration (RfC) for 2,4,6-TNT has not been derived.

Limited information is available on the reproductive or developmental toxicity of 2,4,6-TNT to animals or humans following inhalation exposures. Information from occupational exposure studies suggests that 2,4,6-TNT may cause menstrual disorders and male impotency (Zakhari and Villaume 1978; Jiang et al. 1991).